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## Acute Transverse Myelitis Associated with *Salmonella* Bacteremia: A Case Report

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**Conflict of interest:** None declared

**Patient:** Female, 28  
**Final Diagnosis:** Acute transverse myelitis  
**Symptoms:** Ascending paralysis  
**Medication:** —  
**Clinical Procedure:** —  
**Specialty:** Infectious Diseases

**Objective:** Rare disease





**Background:** Acute transverse myelitis (ATM) is an uncommon and often overlooked complication of certain bacterial and viral infections that can have a rapid onset and result in severe neurological deficits.

**Case Report:** This case report describes a previously healthy 28-year-old woman who presented to the trauma center after developing acute paralysis and paresthesias of all four extremities within the span of hours. The initial presumptive diagnosis was spinal cord contusion due to a fall versus an unknown mechanism of trauma, but eventual laboratory studies revealed *Salmonella* bacteremia, indicating a probable diagnosis of parainfectious ATM.

**Conclusions:** This case illustrates the importance of considering the diagnosis of parainfectious ATM in patients presenting with acute paralysis with incomplete or unobtainable medical histories.

**MeSH Keywords:** Bacteremia • Quadriplegia • Salmonella Infections

**Full-text PDF:** <http://www.amjcaserep.com/abstract/index/idArt/900730>

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## Background

Acute transverse myelitis (ATM) is an inflammatory disease of the spinal cord that presents with motor, sensory, or autonomic neurological deficits below the level of injury [1]. In its acute form, symptoms can progress within hours and worsen over days [2]. Symptoms of ATM generally reach peak severity between 4 hours to 21 days [2,3]. ATM is uncommon, with a historical incidence of 1.3–4.6 per million people, though its underdiagnosis, attributed primarily to its variable presentation, has led to a recent estimated incidence as high as 3.1 per 100,000 patient-years [4–7]. Because of its rarity, ATM can be an overlooked diagnosis, especially in the acute care setting in which it may be an unfamiliar entity to the providers.

Transverse myelitis can be caused by multiple etiologies that include idiopathic, parainfectious, post-vaccinal, and paraneoplastic, or it can be associated with autoimmune diseases like sarcoidosis, systemic lupus erythematosus (SLE), neuromyelitis optica, or multiple sclerosis [1]. Of these etiologies, the parainfectious form is the most common, and well-recognized causal agents include enterovirus, hepatitis A virus, primary cytomegalovirus (CMV), herpes simplex virus, influenza, human immunodeficiency virus (HIV), Epstein-Barr virus, mycoplasma, and *Mycobacterium tuberculosis*. In this report, we present a patient with ATM caused by *Salmonella* bacteremia, an organism implicated in only several documented cases of ATM [1,2].

## Case Report

A 28-year-old African American woman presented to the R Adams Cowley Shock Trauma Center in Baltimore, Maryland, for evaluation of right-sided paralysis and left-sided paresthesias after being found down in her home between her nightstand and her bed. The symptoms began after eating at a local restaurant chain, consuming alcohol, and smoking one cigarette dipped in liquid phencyclidine (PCP). She was asymptomatic that night and went to bed without complications. She was found down next to the bed the following morning unable to

move. The patient denied any physical trauma other than falling from the bed. She had no history of seizures, sickle cell anemia, fever, urinary or bowel incontinence, or pain. The patient had a history of viral meningitis without residual deficits eight years prior, gastric bypass surgery, and cholecystectomy, and she was treated for a presumed urinary tract infection five days prior to admission at an outside institution.

Her triage vital signs were as follows: temperature 36.8°C, blood pressure 155/100 mm Hg, heart rate 90 beats per minute, respiratory rate 16 breaths per minute, and an oxygen saturation of 95% on room air. On physical exam, the patient was alert and oriented to person, place, and time. Her cranial nerves II–XII were grossly intact. She had 2/5 strength in her right lower extremity, 3/5 in her left lower extremity, and 4/5 bilaterally in her upper extremities. Her sensation was intact. She opened her eyes spontaneously, her verbal response was oriented and appropriate, and she obeyed commands, resulting in a Glasgow Coma Scale score of 15. Pupils were equal, round, and reactive to light. She had tenderness to palpation over the cervical, thoracic, and lumbar spine without obvious external signs of trauma. Her anal sphincter tone was intact.

Multiple laboratory abnormalities were noted on admission, including hyperlactemia, elevated liver function tests, and electrolyte derangements. Her complete blood count and remainder of her complete metabolic panel were within normal limits. These values are summarized along with reference ranges in Table 1. Her toxicology screen was positive for PCP. Blood cultures drawn on admission grew the aerobic bacteria *Salmonella enterica* serotype 4,12: i, and then repeat cultures drawn two days later grew group B streptococcus (aerobic and non-aerobic). Subsequent blood cultures were negative, as were stool cultures. Metronidazole and vancomycin therapy was initiated empirically, with transition to ceftriaxone upon learning of the culture results. In addition, she had a detailed autoimmunity workup including anti-nuclear antibody (ANA) and HLA-B27, all of which returned within normal limits.

Admission computerized tomography scan of her cervical, thoracic, and lumbar spine revealed no spinal fractures. Magnetic

**Table 1.** Select serum laboratory results.

Laboratory parameter	Patient value	Reference range
Lactic acid	2.5 mmol/L	0.5–1.6
Aspartate aminotransferase	76 units/L	10–34
Alkaline phosphatase	128 units/L	44–147
Potassium	3.3 mmol/L	3.5–5.5
Total calcium	8.1 mg/dL	8.6–10
Ionized calcium	1.09 mmol/L	1.1–1.35

**Table 2.** Select CSF laboratory and culture results.

Laboratory parameter	Patient value	Reference range
CSF protein	90 mg/dL	15–45
CSF red blood cell count	350/ $\mu$ L	None
CSF IgG	12.7 mg/dL	0.0–8.1
CSF white blood cell count	4 $\mu$ L	0.0–5
CSF glucose	59 mg/dL	50–80
CSF lymphocytes	73%	60
CSF organism growth	None	None
CSF oligoclonal bands	None	None
Neuromyelitis optica/AQP4 IgG, CSF	Negative	Negative

CSF – cerebrospinal fluid; IgG – immunoglobulin G; AQP4 IgG – aquaporin-4 autoantibody.

**Table 3.** Select infectious disease laboratory results.

Laboratory parameter	Patient value	Reference range
HIV	Nonreactive	Nonreactive
HBV core	Nonreactive	Nonreactive
HBV surface	Nonreactive	Nonreactive
Herpes virus type 6 IgM	Negative	Negative
QuantiFERON-TB gold	Negative	Negative
West Nile virus IgG	Negative	Negative
West Nile virus IgM	Negative	Negative
EBV IgG	Positive	Negative
EBV IgM	Negative	Negative
CMV IgG	Negative	Negative
CMV IgM	Negative	Negative
<i>B. burgdorferi</i> IgG	Negative	Negative
<i>B. burgdorferi</i> IgM	Negative	Negative
IgM (peripheral blood)	94 mg/dL	72–126
IgA (peripheral blood)	243 mg/dL	139–261
IgG (peripheral blood)	1158 mg/dL	853–1463
Total immunoglobulin	1450 mg/dL	1104–1810

HIV – human immunodeficiency virus; HBV – hepatitis B virus; TB – *Mycobacterium tuberculosis*; IgG – immunoglobulin G; IgM – immunoglobulin M; EBV – Epstein-Barr virus; CMV – cytomegalovirus; *B. Burgdorferi* – *Borrelia burgdorferi*.

resonance imaging (MRI) revealed no acute intracranial abnormalities, but enhancement within the central aspect of C3–T2 suggested spinal cord edema, disc protrusions at C3–4, C4–5, and C5–6, and bilateral posterior neck muscle edema. A lumbar puncture was not initially performed upon patient admission due to concerns about raised intracranial pressure from suspected trauma.

Four hours after presentation, her strength had progressively deteriorated to 1/5 in all extremities. She lost proprioception in her

toes bilaterally and developed absent anal sphincter tone. The patient was admitted to the neurotrauma intensive care unit with further deterioration ultimately requiring endotracheal intubation due to respiratory failure. Upon her positive blood culture results, lumbar puncture was performed, which demonstrated a pattern consistent with ATM, the results of which are summarized in Table 2. An extensive workup for infectious etiologies was also completed, with the results summarized in Table 3. Plasma exchange therapy was initiated with a suspected diagnosis of ATM.

Despite maximal medical therapy, the patient remains quadriplegic, is experiencing neurologic pain, received a tracheostomy due to prolonged respiratory failure, and required a feeding jejunostomy tube placement at the time of this report.

## Discussion

ATM results from inflammatory spinal cord injury and classically presents with bilateral ascending numbness or weakness of the legs, with the upper extremities generally less severely affected. Signs of autonomic dysfunction are also common, including urinary retention, incontinence, constipation, fecal incontinence, and sexual dysfunction [1–3]. The pathogenic mechanism remains unclear but is conjectured to be due to either molecular mimicry or microbial superantigen-mediated inflammation generating an immunologic response leading to spinal cord inflammation [3,8,9].

The present case describes a woman with prior history of a presumed urinary tract infection, bariatric surgery, and recent PCP use presenting with progressive, ascending neurological deficits resulting from *Salmonella* bacteremia. Increased risk of *Salmonella* infection has been associated with recent antibiotic use and past history of gastric surgery [10]. Bariatric surgery can cause a reduction in secretion of gastric acid while antibiotic use disrupts the natural microbiome of the alimentary tract, together allowing for evasion of natural immunological defenses and creating an environment conducive to non-native bacterial growth [10,11]. Moreover, a preceding viral or bacterial infection prior to onset of symptoms has been reported in up to 38% of patients who develop acute ATM [1]. The Transverse Myelitis Consortium Working Group previously identified working diagnostic criteria, specifically indicating that history of recent previously documented infection in combination with evidence of spinal inflammation on MRI is highly suggestive of the diagnosis of parainfectious ATM [1], although other autoimmune or inflammatory causes cannot be excluded.

PCP works as an NMDA receptor antagonist, and intoxication commonly presents with neurological symptoms including ataxia, amnesia, nystagmus, aggressive behaviors, hallucinations, and even catatonia [12,13]. However, PCP use is not known to be associated with cases of ATM. Interestingly, NMDA receptor activation has been shown to regulate the tight junctions on cerebrovascular endothelial cells [14]. The patient's concomitant PCP use may have altered the permeability of her cerebrovascular endothelial cells, permitting translocation into the cerebrospinal fluid (CSF). This alteration could have allowed for more fulminant penetration of infection and inflammation into her central nervous system (CNS), ultimately contributing to the acute severity of her presentation. Additionally, both PCP

and alcohol are known to cause increases in liver transaminases, as seen in the current case.

In this case, differentiating between possible etiologies for acute myelopathy was approached through clinical history, physical exam, and immunologic and radiologic findings. Diseases like acute disseminated encephalopathy and neuromyelitis optica were dismissed due to the lack of presenting diagnostic criteria, namely, altered mental status or aquaporin-4 (AQP4) immunoglobulin G (IgG) autoantibody [15,16]. Additionally, while this case is typical of a “clinically isolated syndrome” (CIS), with only one lesion and one attack instance, patients with acute complete transverse myelitis only have a cited risk of 5–10% of developing multiple sclerosis [17]. Also, this presentation does not fulfill the McDonald criteria of dissemination within space and time necessary for a diagnosis of multiple sclerosis [18]. Radiographic evidence suggestive of ATM includes spinal cord lesions extending over three segments on T2-weighted MRI images as well as hypointensity on T1-weighted images during acute episodes. MRI imaging also may show the presence of cord swelling and single continuous multisegmental spinal lesions [1,9]. This patient's extensive spinal cord edema extending from C3 to T2, acutely progressive neurologic deficits, and recent history of infection point to ATM as the most likely etiology, according to the current working diagnostic criteria [1].

For diagnosing parainfectious ATM, lumbar puncture is an unreliable indicator as proof of the presence of infectious organisms or antibodies in CSF and can be futile in cases where ATM develops after the initial infection has subsided [1]. Furthermore, a normal CSF leukocyte count does not exclude the diagnosis of ATM. However, lumbar puncture can detect signs of inflammation such as pleocytosis, elevated protein, oligoclonal bands, or elevated IgG that may affirm an inflammatory process, arguing against the presence of a vascular, toxic/metabolic, neurodegenerative, or neoplastic myelopathy that was not recognized on MRI [5]. Indeed, the patient's lumbar puncture was notable for elevated protein concentration, present in about 50% of transverse myelitis patients, as well as elevated red blood cell count and IgG [19]. Thus, the specific MRI findings and characterization of CSF were both needed to rule out other myelopathic etiologies. To date, there are two case reports describing the occurrence parainfectious ATM occurring after *Salmonella* bacteremia [9,20]. In both cases, the patients were women who presented with progressive bilateral lower extremity limb weakness, with a CSF negative for Gram stain and cultures and admission blood cultures demonstrating *Salmonella* bacteremia. The first case report described a woman with recent urinary incontinence with 1–2 weeks of progressive paralysis of her lower extremities. She was diagnosed with ATM secondary to *Salmonella enterica ser. Paratyphi B* [9]. In the second case, a woman with a history of chronic

hepatitis C infection and splenectomy presented with a 3-week history of progressive lower extremity weakness resulting from ATM secondary to *Salmonella* type D nontyphi [20]. Our case is distinct from these previous reports in the rapid onset of symptoms, which occurred over hours instead of weeks, and the bacteria type, *Salmonella enterica serotype 4,12: i*, associated with the onset of ATM.

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## Conclusions

Clinicians should consider the complication of ATM in patients presenting with *Salmonella* bacteremia as prompt detection and treatment could help prevent or even reverse the progression of symptoms. By adding this patient's experience to the growing canon of information regarding ATM, we hope to help improve the timeliness of the diagnosis of these patients in the future.